THE SYMPTOM COMPLEX OF TRANSVERSE LESION OF THE SPINAL CORD AND ITS RELATION TO STRUCTURAL CHANGES THEREIN.

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ELSEWHERE[1] I have discussed the pros and cons concerning laminectomy in cases of fracture-dislocation of the spinal column. In studying the literature on this subject one is struck with the absolute impossibility of being able to determine definitely in many cases, within any reasonable time, whether the spinal cord has been irrevocably destroyed as a conductor of afferent and efferent impulses. Many investigators, chiefly surgeons, hold up the fact that a patient with a fracture-dislocation showing symptoms of complete transverse lesion occasionally will recover in the course of time, regaining a fair amount of power and usefulness in the lower limbs. Upon such a history they base their arguments as to the possibility of regeneration in the spinal cord when it is badly crushed. The next step in such logic is, that if such a patient can occasionally get well without laminectomy how many who die without this surgical procedure would have lived had their spinal canal been opened, clots and spicules of bone removed, and the cord given a chance to bridge with functionating nervous tissue the area of traumatic softening.

Now, let us keep before us this important fact: The neurons of the central nervous system, when acted upon by toxic agents or physically injured by factors either from without (concussion, fracture-dislocation, etc.) or from within (pressure of tumors, exudates, etc.), although structurally damaged, yet are able to exhibit, up to a certain point, an autorestoration, provided the factor or factors causing this condition have been removed or ameliorated.

Literature is not wanting in cases presenting symptoms of grave transverse involvement of the spinal cord the histological examination of which discloses little and insignificant structural changes. Gordon Holmes[2] reports 3 such cases which were the result of the collapse of tuberculous vertebræ with consequent pressure on the spinal cord. A fourth case in the same contribution was one of glioma of the pons. The paraplegia and sensory disturbance were so ex-

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[1] Injuries of the Spinal Cord, Jour. Amer. Med. Assoc., March 21, 1908.

[2] On the Relation between Loss of Function and Structural Change in Focal Lesions of the Central Nervous System, with Special Reference to Secondary Degeneration. Brain, 1906, iv, 514.

treme in the tuberculous cases that one would have been warranted in expecting to find a practically complete destruction of the spinal cord at the level of the bone lesion, with pronounced secondary ascending and descending degeneration. In 2 of the cases the paraplegia was present for about one year before death, in 1 case for five months. The microscopic examination of the segments involved revealed but a very moderate change, and above and below the site of the lesion there was no secondary tract degeneration found by the Weigert or the Marchi method. The fourth case presented at first a right hemiplegia, and with the further growth of the neoplasm a complete paralysis in all four extremities obtained for one month before death. Here, again, the histological examination failed to discover any secondary degeneration.

Holmes argues that the loss of the medullary sheath at a given point is liable to render the axis cylinder non-conducting, but does not necessarily entail the secondary degeneration above or below, as the case may be. Therefore, in such a case one might examine Weigert or Marchi preparations functionally distal to the lesion, and find a normal or almost normal condition of affairs, whereas in reality the tract at the point of injury would be minus the myelin sheaths and not a conductor of impulses. He cites the findings in disseminated sclerosis to support this view. In this disease there is a loss of myelin for a short distance only, with preservation of the axis cylinders, and yet a total break in the conducting ability of the fibers. In other words, he infers that the myelin is necessary to conduction, that its removal does not affect the anatomical integrity of the axis cylinders or its perikaryon. From these premises he draws the conclusion that reparative process would be possible.

This reasoning of Holmes is all that could be desired, until he comes to drawing the parallel between the non-conductivity of bare axis cylinders and disseminated sclerosis. In disseminated sclerosis, although a tract of axis cylinders bereft of their medullary sheaths remains to the end without medullary covering, the symptoms due to non-conduction of impulses in this area of the cord frequently pass away and there is every evidence that the nerve fibers in this area are functionating normally or almost so.

Schlagenhaufer[3] reports a case in which an endothelioma of the dura mater situated at the edge of the foramen magnum pressed upon and grossly distorted the upper cervical segments of the spinal cord and the medulla oblongata. The duration of this growth was one year and four months, and before death the patient presented a complete paralysis of all four extremities, as well as of the bladder and rectum. Complete anesthesia of the four extremities and the trunk was also present. Although there was pronounced change in

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[3] Ein intradurales Endotheliom im Bereiche der obersten Halssegmente, Arbeiten aus dem Neurologischen Institut, a. d. Weiner Universität, 1902, viii, 88.

the second and third cervical segments (alle Stadien einer Compressionsmyelitis), yet secondary degeneration was conspicuous on account of its absence.

Bielschowsky,[4] in a painstaking investigation of axis cylinders when involved in tumor formation or in compression of the spinal cord, also thinks that they have the power to regenerate. His conclusions are interesting when compared with those of Ballance and Stewart[5] who were the sponsors in large measure for the neurilemma-nuclei theory of axis-cylinder regeneration. Bielschowski found in the outer zone of infiltrating tumors of the central nervous system, as well as in the compressed level of the spinal cord, naked axis cylinders which had been interrupted in their course by the pathological process, but were still in unbroken communication with their perikaryons. At the ends of these axis cylinders there were fine fibril-like prolongations ending in a peculiar oval-shaped structure not unlike the terminal organ of Held. On account of their oval shape and light centres he speaks of them as *Ende Ringe.* He considers these long, fibrillar prolongations the products of regeneration. The absence from the equation of neurilemma nuclei is noteworthy, and the fine, newly formed bloodvessels are supposed to exert the chemotactic influence and to prepare the path for the newly formed nerve fiber which clings to its outer surface, showing here the expanded end-bulbs.

Ballance and Stewart[6] say: "The more the specimens are studied the more is the conclusion forced upon the mind of the observer that for the regeneration of a peripheral nerve fiber (not only the axis cylinder, but also the medullary and neurilemma sheaths) the activity of one variety of cell, and one variety only, is responsible. That cell is the neurilemma cell." These investigators further submit that the reasons we do not have any regeneration in the axones of the central nervous system is that in this locality there is an absence of neurilemma nuclei.

Marinesco[7] holds that there is no *régénérescence autogène* in the sense that there occur islets of new-forming axis cylinders and medullary sheaths in the distal part of a divided nerve. He thinks that the new nerve fibers in the distal part are prolongations from growing tissue in the proximal end. In speaking of experimental cases in which nerves were divided and their proximal portions removed, microscopic examination later demonstrating newly formed fibers in the distal portion, he evidently does not consider that he weakens his theory by naively stating: *De pareils cas sont d'autant*

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[4] Ueber das Verhalten der Achsenzylinder in Geschwülsten des Nervensystems und in Kompressionsgebieten des Rückenmarks, Jour. f. Psychol. und Neurol., 1906, vii, 101.

[5] The Healing of Nerves. Macmillan & Co., Ltd., 1901.

[6] Loc. cit., p. 89.

[7] Etude sur le mécanisme de la régénérescence des fibres nerveuses des nerfs périphériques, Jour. f. Psychol. und Neurol., 1906, vii, 140.

plus difficiles à interpréter que les cellules d'origine du bout arraché sont complètement atrophiés.

The raison d'être of this seeming digression from the title of this paper lies in the presentment of the leading thoughts on the subject of nerve regeneration. It makes a considerable difference in the way we view trauma and tumor involvement of the central nervous system if we accept the opinion of Bielchowski. As I have said, we have to admit a capability on the part of the central neuron of autorestoration (not the régénérescence autogène which Marinesco denies); likewise, it seems to me that it is axiomatic that the nerve cells and their processes, axis cylinders as well as dentrites, in the central nervous system must be amenable to the same laws of wear and tear and death that govern cells elsewhere in the body. The large, pyramidal cells of the motor cortex, for example, are limited in their length of life, and when their usefulness is past, phagocytic action removes them and their work is assumed by others. Such a theory presupposes a constant appearance of new neurons. The supply of the perikaryon might be from the development of the many small and insiginficant somatochrome cells with which the gray matter throughout the central nervous system is studded. But the axis cylinder cannot be so easily accounted for unless we accept the idea of the growth from the cell body. The evidence seems to point to the fact that a limited amount of regeneration in the central nervous system is possible after trauma under favorable conditions. But this regeneration is too insignificant to be of functional importance.

Through Dr. Spiller's kindness I have been enabled to study the spinal cord of a case which was in his service in the Philadelphia General Hospital, and which bears interestingly on the question of slight structural changes accompanied by grave symptoms. The case is one of 11 embodied in a paper by Spiller and Weisenburg.[8]

The patient, a woman, aged about forty years, ten months after the removal of her left breast for carcinoma, began to suffer from radiating pains across her chest and back, as well as in the lower limbs. One month after the onset of these symptoms she suddenly became paralyzed in her lower limbs, and was confined to her bed. She complained of a girdle sensation, but did not suffer extreme pain. The upper limbs were not involved. There was no motion whatever in her lower limbs, which were the seat of flexor contractures, the thighs on the abdomen, the legs on the thighs. The patellar and Achilles tendon jerks could not be elicited. Babinski reflex was present on both sides. Sensation for touch and pain was lost in the lower limbs and trunk up to a line three inches below the umbilicus. Occasionally pin prick would be recognized on the dorsum of each foot. Incontinence of urine and feces was present. She died almost two months after the onset of her paraplegia, and therefore sufficient time had been given for secondary degeneration. The diagnosis of vertebral carcinoma in the thoracic region was ratified at the necropsy.

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[8] Carcinoma of the Nervous System and the Report of Eleven Cases, Jour. Nerv. and Ment. Dis., August, 1906.

In studying the spinal cord microscopically, I expected to find very well-marked degeneration and wholesale destruction of tissue through pressure. Sections were made at intervals from the third thoracic segment down to and including the first lumbar segment and stained by the Weigert hematoxylin as well as the hemalum-acid fuchsin methods. To the unaided eye there was none of that yellowish color in the Weigert sections which betokens degeneration. Under the microscope there were seen comparatively few swollen axis cylinders and swollen medullary sheaths in the lateral and posterior columns. There were also found here and there a few spaces from which it appeared that axis cylinders had dropped. There was a moderate number of granular cells. The chief point of interest is that the spinal cord which appeared so little altered even microscopically had in reality ceased to be a conducting mechanism.

I think that we are justified in assuming that had this condition been caused by a fracture-dislocation instead of a rapidly increasing malignant growth, the patient, in course of time, would have shown some return of function, which would have been ascribed by certain observers to regeneration after complete transverse lesion.

It is, indeed, remarkable that the spinal cord can so completely suspend its function, at the same time presenting so slight evidence of organic change. And it should make us particularly careful in giving a positively bad prognosis as to return of function in cases which have presented for some months symptoms of complete transverse lesion.